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Hemodynamic Effects of Strength Exercises

by

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Strength exercises may impose an extremely heavy cardiovascular load due to increased heart rate and a high increase in blood pressure. For this reason, despite its proven efficacy, patients who would benefit from strength training were directed away from it. The magnitude of heart rate and blood pressure increases evoked by strength exercises depend mainly on the ratio between strength developed and maximal strength, and on the number of repetitions of a given exercise. The attitude to this form of training over the last two decades changed to such a point that strength training became recommended even for cardiac patients. Nevertheless adverse effects of strength training have to be taken into account: several cases of cerebral hemorrhage during strength training in apparently healthy young subjects have been registered. It seems desirable to assess individual response of cardiovascular system to strength exercises, to this end it might be useful to better understand the mechanisms of this response. However at present relevant data is scarce and inconsistent. Of special interest is the interpretation of the role of Valsalva effect, which is spontaneously performed while exercising with very heavy loads. This maneuver on one hand adds to the rise in arterial pressure and on the other may create a protection against the rise of transmural pressure acting on heart's and brain arterial vessel walls.

Key words: *strength training, heart rate, blood pressure*

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Strength training

The primary goal of strength training is to increase muscle strength. The basic feature of all kinds of strength exercises is to move the body's musculature against an opposing force, called resistance, hence such training is alternatively termed resistance training.

A repetition is the basic unit of strength training. Usually a repetition consists of the concentric phase, when the active muscles shorten, and the eccentric phase, when they lengthen.

Repetitions performed continuously without stopping are termed a set. The number of repetitions in a set usually ranges from 1 to 15. The load, or resistance, used may be expressed in absolute units, as percentage of maximal force which can be developed voluntarily, commonly however is expressed as the maximal number of repetitions, which can be performed without stopping and therefore is termed repetition maximum (RM). The greatest resistance is defined as such that only one repetition can be performed and is denoted by 1-RM. The lower the resistance, the more repetitions can be performed. For instance using 85% 1-RM usually 3 repetitions can be performed thus 85% 1-RM equals 3-RM, and 80% 1-RM equals 8 RM, as at this resistance maximally 8 repetitions are typically possible. Expressing intensity with maximal number of repetitions (RM) refers to current capabilities of a trained individual, thus for example 1-RM means different resistance for different subjects. If, due to training, muscle strength increases maintaining constant RM requires increasing absolute resistance.

The intensities from 1-RM to 6-RM are the most effective in increasing strength, whereas intensities above 25-RM are marginally effective or ineffective (Atha 1981). Thus it is not possible to replace several repetitions performed against heavy resistance with larger number of repetitions performed against light resistance. Intensity of 50% 1-RM suffices to increase muscle strength, 80% 1-RM may be indispensable to increase bone mineral density (Vincent and Braith 2002).

Strength training as a therapy

Beside body-building and weight-lifting, where strength exercises are the main aspect of training, such exercises are performed in many other sport disciplines, constituting part of the training program. Strength training also established its position in recreational activity. Resistance programs have proven itself as an efficient therapy against age dependent loss of strength and muscle mass (termed sarcopenia) and against muscle weakness due to illness induced

inactivity. Maximal muscle strength declines gradually, by about 15% per decade between the age of 50 and 70 years. Later in life the rate of loss doubles, so subjects past 70 retain half or less than half of their peak strength (Doherty 2003). This age dependent loss of maximal strength, reduces the strength reserve and brings elderly persons towards their threshold for dependence (Young and Skelton 1994, Rantanen and Avela 1997, Kozakai et al. 2000), and increases the risk of falling (Morley et al. 2001).

Resistance training may at any age reverse sarcopenia, at least partly. An increase of over 100% in knee extensors 1 RM was reported by Fiatarone et al. (1990) in a group of 72-98 year old men and women after 10 weeks of resistance training. Even a greater increase (over 130%) was noted by Harridge et al. (1999) in a group of elderly subjects (85-97 years) after 12 weeks of strength training. Smaller improvements, ranging from 26 to 50% of knee extensors 1 RM, were reported in men and women in their 7th and 8th decades of life (Charette et al. 1991, McCartney et al. 1996, Hakinnen et al. 1998, Hunter et al. 1999, Tracy et al. 1999, Yarasheski et al. 1999, Hagerman et al. 2000, Lemmer et al. 2000, Brose et al. 2003 Ferri et al. 2003). Roelants et al. (2004) and Reeves et al. (2004) found improvements below 20%. The reasons for such diversified results according to Reeves et al. might be: average age of participants, pre-training status, neural adaptation occurring during familiarization period resulting in increased weight-lifting capacity, volume and intensity of training protocols.

The beneficial effects of resistance training on bone density are superior in comparison to endurance training, however with respect to maximal oxygen consumption, blood pressure, lipid profile, hypertension, obesity inferior (Pollock and Evans 1999).

A training session includes all exercises performed in a given time period. A recommended training session for older adults consists of 8 exercises, which increases strength of large muscle groups that are important in everyday activities (arms, shoulders, spine, hips, and legs). An intensity equal to 80% 1-RM (i.e. 10-15 repetitions per set) seems to be the recommended amount of load necessary to produce significant changes. At the beginning of a training program it is suggested to perform only one set of each exercise with 2-3 min rest periods between sets. Progression can go from 1 to 3 sets over training time for each type of exercise. The training consisting of 2-3 session per week is recognized as sufficient for gaining health benefits, each session consisting of single sets of 8-10 exercises (Evans 1999, Fleck and Kraemer 1997). Similar recommendations can also be found in (ACSM 1998, ACSM 2002, Feigenbaum and Pollock 1999, Mazzeo and Tanaka 2001).

Cardiovascular safety during strength exercises

Strength training is often recommended with caution due to the belief that it will be accompanied by a large rise of arterial blood pressure. Indeed, during different forms of strength training high increase in blood pressure and heart rate has been observed (Fleck and Dean 1985, MacDougall et al. 1985, Fleck and Dean 1987, Stone et al. 1991, Sale et al. 1993, Sale et al. 1994, Scharf et al. 1994). However, a relative safety of strength training has been claimed (Sheldahl et al. 1983, Sheldahl et al. 1985, Vander et al. 1986, Keleman et al. 1986, Crozier et al. 1988, Sparling i Cantwell 1989). Is the claimed cardiovascular safety of strength training justified? Rather, it seems more appropriate to speak about similar challenge when comparing cardiovascular produced by strength versus endurance training [Vinson et al. 1990, Faigenbaum et al. 1990, Green et al. 2001]. Vinson et al. observed in some subjects the maximal response to resistance exercise being less than to endurance exercise, while the reverse in others. Faigenbaum et al. observed much more adverse cardiac events in cardiac patients during the graded exercise stress test than during maximal contraction effort or during repetitions with submaximal force.

Hemodynamic effects of strength exercises

MacDougall et al. (1985) described basic features of the pressure response to strength exercises: rhythmical changes of the arterial pressure in accord with concentric and eccentric phases of exercise and very high amplitude of these changes. They also found that immediately following the last repetition blood pressure declined below pre-exercise level. These principal features of the circulatory response to a series of strength exercise were confirmed in subsequent studies though detailed pictures differed in many points. Already in this study conflicting data are presented regarding changes of nadir values of blood pressure. These authors measured blood pressure invasively in five experienced body builders during single-arm curls, overhead presses and both double- and single-leg press performed to failure at 80, 90, 95 and 100% of 1-RM. In their study systolic and diastolic pressure rose rapidly during the concentric phase and declined rapidly during the eccentric phase. Two traces presenting continuos blood pressure records from two subjects performing a leg press at 90% 1-RM show that nadir pressure remains slightly below baseline during the whole exercise protocol. However schematic drawing illustrating mean blood pressure during leg press at 95% 1-RM shows continuous rise of the nadir pressure almost to the end of the exercise protocol. The peak pressure grows along subsequent repetitions both in blood pressure traces as well as in the figure

presenting time course of mean blood pressure changes. Highest values of peak blood pressure were attained during the double-leg press, reaching an average of 320/250 mmHg. Substantially lower peak pressure was measured during the single-arm curl (255/190 mmHg), while peak pressure observed during the single-leg press was between the former two. In this study the authors attempted to elucidate the role of Valsalva maneuver in enhancing instantaneous pressure rises when performing repetitions of strength exercise. They observed that this maneuver was performed with heavy lifts at 1-RM or during latter repetitions with lighter loads. They noted that mouth pressure never exceeded 70 mmHg. The authors did not provide any data, which allows to determine the contribution of the intrathoracic pressure to aortic pressure rise during exercise. Nevertheless they supply interesting data relating to mouth pressure in the rise of arterial pressure at rest: they found that the average mouth pressure of 135 mmHg elevated arterial pressure from 135/90 to 190/170 mmHg.

In a subsequent study MacDougall et al. (1992) provided data showing the magnitude of the intrathoracic pressure generated by Valsalva maneuver. Intrathoracic pressure was determined indirectly by measuring pressure in the esophagus, while blood pressure was measured invasively. They found that Valsalva maneuver only occasionally assists exercises when force output remains below 80% of MVC. With heavier loads this maneuver becomes almost inevitable. They also confirmed their earlier observation that even if Valsalva maneuver with lighter loads is absent at the beginning of a set, it appears closer to the series end of it, if the exercise continues to failure. In this stage of series subsequent intrathoracic pressure progressively rose, as well as rises of both systolic and diastolic pressure.

They extended their observation regarding the effects of various intrathoracic pressures on arterial blood pressure at rest. However the inconsistent description of the results makes their interpretation very difficult. In the figure showing the relation between intrathoracic pressure (x axis) and arterial pressure (y axis) the x axis scale represents intrathoracic pressure in Torr, ranging from 20 to 100 mmHg. However the legend for this figure states that x axis is scaled in percentage of maximum voluntary Valsalva pressure. According to a latter description, absolute values of the intrathoracic pressure are not presented, thus it is impossible to relate the rise of intrathoracic pressure to the rise of arterial pressure. At rest, i.e. when no Valsalva was performed, blood pressure was approximately 130/85 mmHg. When this maneuver was performed at lowest intrathoracic pressure, blood pressure was about 210/130 mmHg, and rose continuously with rising intrathoracic pressures, reaching finally a value of 280/190 mmHg. Interestingly, arterial pressure during the highest intensity (87.5

% MVC) of concentric leg press exercises was below the highest pressure observed during the Valsalva maneuver at rest.

It is impossible to assess to what extent intrathoracic pressure increased blood pressure, as the authors did not compare blood pressure rises when performed exercises with and without Valsalva maneuver at the same force output. Nevertheless they presented interesting data on blood pressure and peak intrathoracic pressure. Both arterial and intrathoracic pressure rose with increasing force output and interestingly the rise of the aortic pressure roughly corresponded with rise in intrathoracic pressure. With only concentric efforts (eccentric phase was performed by assistance) peak blood pressure rose along with the relative load, expressed as percentage of MVC: being 200/105, 241/136 and 263/142 mmHg at 50, 70 and 87.5% of MVC. As peak intrathoracic pressure increased, reaching values of 17, 49 and 51 mmHg respectively, it contributed to the rise in blood pressure. It could be seen from the comparison of blood pressure and intrathoracic pressure rises, that this contribution might constitute only a part of the whole blood pressure increase. Similar values were found with eccentric efforts.

The authors argue that the magnitude of blood pressure response depend mainly on relative intensity of the effort. This is confirmed by the following observation: weight lifting performed by different subjects at the same relative intensity produced similar rise of blood pressure despite individual differences in muscle size or absolute strength. This thesis was further supported by the fact that despite much greater force produced during eccentric effort than during concentric one (about 50%) blood pressure rise was similar, if relative strength was similar. The decisive role of relative strength is confirmed by the observation, that blood pressure peaks occurred when the knee joint angle was 90º, during the beginning and final position of lifting the weight. Blood pressure nadir coincided with the 170º knee joint angle, at which there was an extension or a "lock out". The authors argue that a relative force was greatest with knee flexed, smallest with extended, because the former position is the weakest position in the strength curve, the latter the strongest. As the absolute force output is the same during the whole exercise, the muscles require greater stimulation when knees are flexed, less when they are extended, thus the relative load is greater in the former position, smaller in the latter. The authors strengthen their reasoning by demonstrating that during isometric contraction at different knee joint angles, blood pressure rise was similar when relative effort was similar. This similar effort was achieved with growing absolute strength, which was greatest with legs extended. This implies that with similar absolute strength relative effort is greatest with flexed legs. It is interesting, and

somewhat strange, that the authors did not consider other explanations, consistent with their previously mentioned observation, that during the concentric phase of a repetition the same force output requires more stimulation then during the eccentric one. In such a case during the concentric phase relative effort will be greater than during the eccentric phase and this might explain the observed increase during lifting and decline during lowering.

Further confirmation of the role of the magnitude of relative effort as the main determinant of blood pressure rise is provided in the study of McCartney et al. (1993). The authors examined changes in HR and blood pressure (measured invasively) in older males (mean: 66 years of age) during 10 repetitions of either single-arm curl, single-leg press or double-leg press performed at 80% and 60% of 1-RM before and after 12 week of strength training. It is not clear, how the values of systolic and diastolic blood pressure as well as heart rate for particular repetitions were calculated, whether these were peak or mean values in this repetition. There is no mentioning of presence of the Valsalva maneuver during the exercises. The rise in blood pressure and increase was significantly greater with 80% 1-RM intensity than with 60%, and was greatest during the double-leg press, while lowest during the single-arm curl, however these differences insignificant. Interestingly, the latter observation is not compatible with the dominant role of relative effort notation. However this data is supported by the results originating from the comparison of the pre- and post-training cardiovascular response to strength exercises. The effectiveness of the strength training program was evaluated by comparing the response to exercise performed with the same absolute strength and with the same relative effort before and after training. Results of such a comparison unequivocally demonstrated the dominant role of relative effort in cardiovascular response in determining the magnitude of cardiovascular response. This response was almost identical to the pre-training one in case of the same relative effort, and significantly weaker in case of the same absolute strength. Of importance for understanding the mechanisms of pressure rise is the concomitant higher rise of pressure and heart rate.

Sale et al. (1993) showed that during a double-leg press blood pressure declined during the lifting phase and rose during lowering. It is evident, that changes of blood pressure followed partly those of intrathoracic pressure. As the subjects started Valsalva maneuver simultaneously with beginning the lifting phase, inthrathoracic pressure rapidly rose elevating arterial pressure, this is visible as a rapid jump in arterial pressure. This rapid rise was followed by prompt intrathoracic pressure decline, which probably caused arterial pressure decline. This decline coincided with leg elevation. Arterial blood changes could

not be explained entirely as an effect of intrathoracic pressure changes. The arterial pressure rise is clearly seen during lowering phase and further and more rapid rise of pressure is superimposed upon this rise. The above description shows the complexity of pressure response arising probably from superposition of at least two processes, each influencing arterial pressure. The rise of arterial pressure during the lowering phase and its decline during the elevating phase was reported by MacDougall et al. (1992). A reverse of this phenomenon, a rise in pressure during elevation and its decline during lowering has been described in an earlier study of MacDougall et al. (1985).

The study of Lentini et al. (1993) was designed to elucidate changes in parameters describing left ventricle function during leg-press exercises. In our opinion this paper rather adds confusion than explains left ventricular response to such exercises. The authors found a significant rise of both systolic and diastolic pressure during the lifting phase. Once the subject achieved maximal leg blood pressure, what has been termed a lockout phase, blood pressure dropped almost back to baseline. It rose again during leg lowering (lowering phase). At this point the description of blood pressure changes ends. It remains unanswered what follows: in our opinion blood pressure achieves second nadir once lowering phase was completed, i.e. when legs were maximally flexed. If this was not the case, how then blood pressure could rise during the lifting phase. If such description is correct, it follows that during every repetition there were two blood pressure peaks interspersed with blood pressure nadir at lockout phase and flanked with blood pressure nadirs during maximal leg flexion at the beginning and end of a repetition. Such description is strikingly different from that presented in earlier studies of this group. As the authors did not present traces of knee joint angle change, it is very difficult to accept or reject such a description. The tracing of arterial blood pressure and intrathoracic pressure presented in this report unequivocally shows that blood pressure changes mirror that of intrathoracic pressure both with respect to time and amplitude. Thus, the question should be asked whether the changes described by the authors pertain to effects of leg muscle activity or rather to the effects of Valsalva maneuver These changes encompass heart rate, left ventricle end-diastolic volume (EDV) and end-systolic volume (ESV) and its derivative stroke volume (SV = EDV - ESV). These authors found a significant decrease of both EDV and ESV during the lifting phase, their recovery during the lockout phase, and their decline during the lowering phase. As a consequence of unidirectional changes in EDV and ESV stroke volume changes were less significant though similar in the direction to those of EDV and ESV. HR was greater during exercise than at rest, however the changes between phases of exercise were small and insignificant.

As a result cardiac output (CO), being the product of HR and SV (i.e. CO = HR x SV) was increased during exercise in comparison to rest, but paradoxically greatest during the lockout phase when blood pressure was lowest, thus the direction of CO changes would rather cause the blood pressure changes in the opposite direction than actually observed. As a mathematical consequence, total peripheral resistance (TPR), which is in fact ratio of mean arterial pressure to cardiac output (i.e. TPR = MAP/CO) showed rather unexpected behavior. It was in comparison to baseline, significantly greater during lifting phase, equal to baseline during lowering phase and less than baseline during lockout phase. No explanation was provided for these findings. As a matter of fact such rapid changes of TPR (50% decline/increase within few seconds are hardly compatible with the slow speed of smooth muscle relaxation and contraction). Moreover, if blood pressure was directly affected by intrathoracic pressure, arterial pressure changes could be unrelated to the true changes in TPR.

Of special interest is the authors consideration of the intrathoracic pressure as protection against heart hypertrophy, needed to generate high blood pressure during strength exercises. The authors argue that transmural pressure i.e. effective pressure acting on the heart wall is diminished due to the countering effect of intrathoracic pressure. It has to be remembered however that Valsalva maneuver both increases arterial blood pressure and at the same time decreases transmural pressure required to produce this increase.

The study of Olivier et al. (2001) demonstrates that pressor response to strength exercises is both of non-neural and neural nature. The authors took the opportunity to examine such response in heart transplant recipients. They studied 3 groups of patients: Early: 3 months post transplant, Intermediate: 1-3 years post transplant, and Late: 5-14 years post transplant. The degree of reinnervation in these groups was probably different; it is known that such reinnervation occurred in patients over 3 years post transplant. The degree of reinnervation might be inferred from the magnitude of heart rate acceleration in response to single leg-press exercise performed by these subjects. The acceleration was smallest in the Early, higher in the Intermediate, and greatest in the Late group; respectively about 5, 11 and 16% increase of heart rate from rest to peak during a single leg-press. Cardiac output was measured by thermodilution, stroke volume was calculated by dividing cardiac output by heart rate. It was found that with respect to upright position stroke volume increased and that percent increases were similar in all 3 groups: 26, 31, and 33% in Early, Intermediate and Late groups respectively. As a result percent increases in cardiac output were clearly different between groups: 30, 40 and 54 %. These differences were not transmitted into similarly high increases in mean arterial pres-

sure (12, 22, 22%) which may result from a decline in systemic vascular resistance (- 18, -18 and -21%). This study demonstrates that pressor response is partly of the non-neural nature, especially increase in stroke volume may depend on Frank-Starling mechanisms. The study has some drawbacks: the thermodilution method does not allow to measure stroke volume beat-to-beat, thus it was impossible to relate changes in SV to exercise phases, furthermore no clear description is provided as to how the variables were calculated. The authors instructed subjects to avoid performing breath-hold maneuvers, however they did not control whether the subjects complied. Therefore this study gives no clues as to what causes rises and declines of blood pressure during single strength exercise, also the effect of Valsalva maneuver can not be ruled out.

Relative effort may not be the only determinant of cardiovascular response during strength exercises. Fleck et al. (1987) compared hemodynamic responses of one-arm overhead press and one-leg knee extension continued to voluntary fatigue at 1-RM (by definition only one repetition), 90, 80, 70, and 50% of 1-RM performed by body builders, novice weight-trained individuals, and sedentary controls. Blood pressure was measured invasively; intrathoracic pressure was not monitored. In order to prevent the performance of Valsalva maneuver the subjects were instructed to inhale during the concentric phase of exercise and exhale during the eccentric phase, yet especially during 1-RM or 90% of 1-RM exercises some subjects showed indications of a Valsalva maneuver. The authors reported only data concerning peak blood pressure, systolic and diastolic and peak heart rate, neither detail describing the eventual evolution of blood pressure nadir nor heart rate changes during the exercise are presented.

Both peak blood pressure and heart rate attained their highest values during the last few repetitions. At the same relative load body builders showed the least response, whereas novice weight-trained and sedentary subjects responded similarly. Significantly weaker hemodynamic response was observed in body builders at the same relative load and much greater absolute load (almost double of that applied in sedentary subjects). Both peak blood pressure, as well as peak heart rate was lower in these subjects. This contradicts the notion that it is only the relative load which determines hemodynamic response to strength exercise. The authors presented no experimental evidence, which explains this response, but they offered three alternative explanations. For instance that body builders used more slow-twitch fibers, what according to some researchers would evoke a weaker pressor response. However such a hypothesis was contested by others and moreover it was also shown, that the percent of such fibers in the arm and quadriceps muscles of body builders is not different than that of sedentary subjects. Other explanations encompass desensitization

of sympathetic nervous system and the resetting of baroreceptors reflex caused by strength training. Also another observation seems to contradict the dominant role of the relative load in determining the magnitude of pressure response. The smallest rise was observed at 1-RM, whereas the response to 90, 80, 70 and 50 % of 1-RM were greater and similar in magnitude. This lack of relative load magnitude effect might be misleading, because the cessation of exercise resulted from voluntary fatigue. The authors did not present the data regarding the number of repetitions performed to fatigue at relative loads used. It is likely, that the number of repetitions was smallest at 90 and greatest for 50% of 1-RM exercises. This might mean that at the point of fatigue the stimulation of muscles were in all cases maximal, thus the relative effort was at this moment similar too.

One more point deserves attention: the maximal values of peak systolic and diastolic pressures were lower than those reported for instance by MacDougall et al. (1985). The greatest peak systolic pressure observed in the group of novice weight-trained subjects was 197 mmHg. As previously mentioned greatest peak blood pressures were significantly lower in body builders (about 150 mmHg) which contrast strongly with values presented by MacDougall et al. (320 mHg).

It seems certain, that avoiding Valsalva maneover while performing strength exercise will significantly limit blood pressure rise, thus will contribute to cardiocascular safety. Evans (1999) states clearly: "With proper breathing technique, the cardiovascular stress of resistance exercise is minimal". However the role of Valsalva maneuver may not be as negative as believed. Haykovsky et al. (2003) stated that contrary to widely held belief heavy strength training will not produce necessarily cardiac hypertrophy of concentric type (i.e. increase in left ventricle wall thickness with minimal alteration of internal cavity dimensions). Nearly 40% of all athletes, who perform strength training have normal geometry of left heart ventricle. In the remaining 40%, for instance in Olympic weightlifters, one can observe concentric hypertrophy, and at rest - eccentric hypertrophy. The authors suggest, that the lack of heart hypertrophy in some athletes might be explained by the protective effect of increased intrathoracic pressure. This pressure, which adds to the tension of heart walls, reduces actively acquired tension needed to produce aortic pressure. However, as already pointed out, one has to bear in mind, that the Valsalva maneuver itself increases arterial pressure. Thus it is possible that protective effect of intrathoracic pressure may be offset by the Valsalva induced rise in arterial pressure. It has to be considered, that all those arteries, which are not compressed by intrathoracic pressure (for instance limbs, neck) will be exposed to the full extend to augmented arterial pressure. Of note, it may be suggested that brain arteries may

also be protected by the Valsalva maneuver. Also arteries in contracting muscles are protected against high arterial pressure.

Haykowsky et al. (2001) examined the left ventricle function during leg-press performed with a brief Valsalva maneuver. They measured central arterial pressure, intrathoracic pressure, and analyzed echocardiographicaly systolic function and wall stress of left ventricle. They found, that despite strong increase of left ventricular end-systolic pressure from 120 mmHg to 255 mmHg at 95% 1-RM, left ventricular end-systolic transmural pressure, calculated as the difference between left ventricular end-systolic pressure and intrathoracic pressure increased much less: from 118 mmHg to 143 mmHg. This modest increase was due to a rise of intrathoracic pressure from 2 mmHg to 112 mmHg. Moderate rise of transmural pressure might explain the lack of significant increase in (calculated) left ventricular end-systolic wall-stress and left ventricular fractional area change (i.e. difference between end-diastolic and end-systolic cavity area divided by end-diastolic cavity area). No significant increase in estimated wall tension might mean that true heart work is much smaller than that deduced from the double product (i.e. HR x SBP), thus hypertrophy inducing stimuli are actually significantly weaker. This in turn may explain why many strength athletes may not develop heart hypertrophy. It has to be stressed, however that the notation of protective role of short Valsalva maneuver performed during strength exercises could be misleading, because as a matter of fact, the Valsalva maneuver protects heart in the first line against itself. True protective role of this maneuver would be possible if the following condition was fulfilled: the rise of intrathoracic pressure caused by the Valsalva maneuver has to be greater than the rise of blood pressure induced by Valsalva maneuver, because only than the transmural pressure will be reduced. This rule in case of strength exercise translates into the following condition. Transmural pressure will be attenuated only if the additional rise of blood pressure (i.e. increase above the level induced by the strength exercise performed without Valsalva maneuver) caused by the same exercise performed together with this maneuver will be smaller than the rise of intrathoracic pressure caused by this maneuver. It is impossible to answer this question on the basis of this study, i.e. assess whether Valsalva maneuver was indeed protective, because the authors did not measure blood pressure during strength exercises performed without the Valsalva maneuver.

Haykowski et al. (2003) addressed yet another crucial risk of strength training: the risk of cerebral hemorrhage. Indeed, several such incidents are reported. By analogy with the possible protective role of the intrathoracic pressure with respect to the heart, protective role with respect to cerebral arteries

might be played by intracranial pressure. This pressure by compressing from outside the cerebral artery will reduce the transmural pressure stretching the vessel, thus will reduce the risk of its rupture. As in the case of heart and intrathoracic pressure, if the Valsalva maneuver is to protect cerebral arteries more than merely against itself, the rise in intracranial pressure has to be greater than the rise of arterial pressure induced by this maneuver. In order to answer this question the authors performed two experiments: they measured invasively the intracranial pressure and non-invasively arterial pressure during the Valsalva maneuver performed at rest and during strength exercise performed without the Valsalva maneuver. They found that the Valsalva maneuver increased intracranial pressure by 18 mmHg and did not change the systolic pressure, whereas during strength exercise intracranial pressure rose by 3 mmHg and arterial by 12 mmHg. Accordingly in the former case cerebrovascular transmural pressure declined by 18 mmHg, in latter rose by 9 mmHg. The authors hypothesized, using data from the literature, that performing Valsalva maneuver during heavy strength exercise will reduce cerebrovascular transmural pressure by 11%. They reasoned that cerebrovascular transmural pressure will be 220 mmHg without the Valsalva maneuver if systolic pressure is 224 mmHg. They estimated that the additional performance of this maneuver would attenuate cerebrovascular transmural pressure down to 198 mmHg, because it would increase intracranial pressure by 64 mmHg and arterial pressure by 37 mmHg. One of the premises is that the rise in intrarthoracic pressure is transmitted 1:1 into a rise of intracranial pressure. As is the case of the previously mentioned study, only comparison of exercise performed with and without Valsalva maneuver can convincingly either confirm or negate this hypothesis.

Conclusions

Strength training may be seen as an effective therapy against sarcopenia in elderly and as a necessary part of patient rehabilitation when due to immobilization or periods of physical hypoactivity muscle strength and mass decline. The efficacy of such therapy is high only if the relative effort is not below 50 % of maximum. Performing strength exercise at such level of force output will inevitably cause significant, often very high, rise of arterial blood pressure. Performing the Valsalva maneuver often accompanies strength exercise, especially at higher loads or when the trainee is fatigued and rises additionally and substantially blood pressure. Blood pressure elevation remains a major concern when recommending strength training, especially in light of very rare, however existing risk of cerebral aneurysm rupture or subarachnoid hemorrhage. For strength training to be effective as a therapy, it must be performed 2-3 times per

week for about 20 min per session. Thus the exposure to the increased cardiovascular load is relatively short and of main concern remains the potential adverse effect of high pressure during exercise. The presented here review of studies devoted to describe the cardiovascular response to strength exercises and to elucidate underlying mechanism shows the scarcity of precise and unequivocal information. There is no uniform data of cardiovascular response; it is even uncertain during which phase of exercise blood pressure rises and when it declines. Unknown are determinants of the magnitude of cardiovascular response, with magnitude of relative effort being the main but not only candidate. It seems likely that much of the confusion is caused by the intervening effect of the Valsalva maneuver, which in some studies was even unnoticed. There is no coherent picture of the mechanism underlying cardiovascular reaction. Likely is the contribution of both non-neural as well as neural mechanisms, such as increased stroke volume and heart rate, however no convincing reconstruction of cardiac and vascular events explaining observed changes in blood pressure has been offered. Of practical importance is whether performing the Valsalva maneuver adds to the cardiovascular risk. It is possible that this maneuver known to increase additionally the rise in blood pressure may also shield against self-incurred hazard or even to provide additional protection, however such hypothesis is at present only speculative. It is evident that at the present state of knowledge warrants further research aimed towards precise description of cardiovascular response to strength exercises and thorough analysis of data to determine the pressor response and explaining the way such response is generated.

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